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THE CEREBRAL ELEMENT IN THE REFLEXES, AND ITS RELATION TO THE SPINAL ELEMENT.*

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The acceptance of the brain as playing an active part in the production of the reflex has been perhaps hampered by theories regarding the various levels involved. The purpose of this paper is, after briefly reviewing the principal steps in the discussion, to present a practical working hypothesis. We hope it may aid in paving the way for a theory which shall embody the results of extended observations unhampered by the old instruction, instruction based upon the supposition that the human reflex is practically that of the frog, instruction the simplicity of which renders it attractive but the application of which leads the student into difficulty.

It is no wonder that authorities are not yet united on the seat of the (so-called) reflex mechanism when they are still at variance on such a fundamental proposition as whether in fact, loss of reflex is the rule or the exception in recent apoplexy and allied conditions, in which the cerebral influence is cut off. We shall cite from our own recent experience a series of cases which tend to show that immediate lessening and loss of reflex in the paralyzed part is the rule, as Pandi stated in 1895, in apoplexy and allied states.

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Prior to the observation of Bastian and others that abolition of the reflexes followed complete destruction of the upper cord, the theory of their mechanism and seat was a simple one. They depended, namely, upon the integrity of a spinal arc, were inhibited by the brain through the pyramidal fibres, and became exaggerated upon the withdrawal of this inhibition.

This simple theory, deduced many years ago from observations on the frog, and applied practically unmodified to the complex human organism, long served for purposes of instruction, and for the explanation of reflexes disordered by disease. Among the ingenious applications of the theory was the explanation of lessened reflexes from cerebral disease as due to destruction of still higher centres which inhibited the inhibition of the reflexes (Gowers). Such suppositions must be abandoned when it appears that the reflexes disappear when the cord is entirely severed from the brain, and thus freed from inhibition as well as from inhibition of inhibition.

The suggestion that shock to the spinal centers may explain the lessened reflexes in cerebral disease has been opposed by Stewart and Turner, Pandi and others. The following fact, which we have repeatedly verified, tends to eliminate shock. Given a case of apoplexy or of broken neck in which the reflexes are abolished and it will be found that the electrical reactions are quite unaffected, until the gradual lessening of electrical irritability which Shirres finds occurs about eight days later in the cases resulting unfavorably. This shows that the mechanism presiding over the electrical reaction is unaltered at a time when that presiding over the reflex* is placed entirely out of commission. If shock has destroyed the activity of the one it should surely to some extent at least affect the other.

The next step was to assume that the so-called centers of the reflexes had ascended in man to the brain (Crocq. Grasset, *et al.*). Crocq, in explaining the varied alterations of superficial and of deep reflexes in disease, went so far as definitely to place the centers for the superficial reflexes in the cortex, and for the deep at the level of the red nucleus. Grasset's modification of this theory assumed that there are in man at least three regions, namely, spinal, basilar and cortical, all of which are possessed of centers for tonicity and reflex, all normally in play in the healthy adult, the higher supplementing and controlling the lower more and more as the animal scale is ascended, though not absolutely replacing them as claimed by Crocq.

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*Without committing ourselves to the acceptation of true reflex element in these phenomena, in fact rather favoring the view that they are merely exhibitions of tonus, we use the current term for convenience.

It was certainly a step in the right direction to credit the brain with playing a part in the production of the reflexes, instead of merely inhibiting them, but Grasset failed satisfactorily to explain the exaggerated reflexes of pyramidal disease.

Already, in 1895, Pandi[1] credited the cortex with being the seat of the entire reflex mechanism, both deep and superficial, explaining the difference between them on the ground that the simplest and most easily excited (the deep reflex) became more prompt through frequent repetition. He quotes Jendrassik and Geigel as having in 1886 and 1892, respectively, determined the cortical origin of the skin reflexes based on clinical and experimental evidence. He explained the increased reflex of old hemiplegics by interference with the spread of the excitation among the association fibers thus leaving the main path to work more freely. He explains the hypertonia, contractures and allied phenomena in the same manner and regards the tendon reflex as merely a sign of increased cortical tonus.

But it is hard to reconcile the pathological variations of the reflexes with this theory. For example, after hemiplegia, when the skin reflexes return they should, by his assumption, become active, like the deep reflexes, whereas they are more faint and sluggish than ever, showing that their control is entirely different from that of the deep reflexes. Again, Pandi's theory hardly explains the increased deep reflexes which result from local disease of the pyramidal tracts. In this case the association fibers of the cortex are unaffected, so that the reflex should be deliberate and rather lessened than increased by the impaired transmission.

In a recent contribution Pandi takes the view that the lesion in case of increased reflexes is irritative and that in a non-irritative lesion impeding the tract the reflexes are lessened. This would seem a difficult proposition to establish, and his position is here open to comment that the skin reflexes should also be exaggerated in irritative lesions, which is by no means the case.

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[1] Pandi: "Der corticale Mechanismus der Reflex-phänomen." Arch. f. d. des Physiol., 1895, LXI., 465-474; also Neurologiches Centralblatt, No. 10, 16 Mai., 1905.

The question has been reopened by Rose[2] who draws his conclusions from the study of the various theories, from experiments on animals and upon extended clinical observations. This author postulates a longer arc with its controlling center in the brain. In his conclusions he assumes either that the brain centers exercise a stimulating action on the ganglion cells of the cord, or that the normal tendon and skin reflexes make use of the longer paths, and that if protective reflex movements and plantar reflexes are retained in disease of the brain, they make use of the shorter paths. He is apparently still influenced by the old teaching that a reflex must pass through, and be controlled by, a single arc. His hypothesis makes no provision for a combined control with varying preponderance of one or the other element.

Such combined control seems to us not only to explain best the variation in the healthy reflex and its alterations in disease, but to fall in line with the modern view of the co-ordinated functioning of the central nervous system in general.

The following comparatively simple proposition seems the logical outcome of the line of thought we have followed. *Both the brain and the cord must play a part in the production of the deep reflexes. The higher arcs tend to produce a more subdued and deliberate reflex, while the lower arcs tend to produce an active, instantaneous and violent reflex. In health the resultant is a comparatively moderate reflex, varying in different individuals as the cerebral or the spinal type predominates. In organic disease the partial withdrawal of the cerebral influence causes the spinal type of reflex, namely the exaggerated reflex. The spinal arc is, however, incapable of alone sustaining the function as shown by the fact that complete withdrawal of cerebral influence causes abolition of all reflexes.*

It is now generally agreed that sudden destruction of the upper cord abolishes the reflexes. There is still some difference of opinion on the question whether upon the *gradual,* complete withdrawal of cerebral influence reflexes may not be preserved as claimed by Raymond and Cestan.

[2] These de Paris, 1905.

The early appearance of the Babinski reflex in broken back when other reflexes are abolished (Stewart and Turner, Walton) has led one of the writers to allow that this reflex was purely spinal. But in a recent case (case 12) of crush of the cervical cord with abolition of all reflexes during the two days of life, the Babinski failed to appear. This observation has been verified in a number of instances by Shirres. Such observations would suggest that even this reflex requires some degree of cerebral influence. This may furnish an important aid in the diagnosis of complete from partial crush of the cord.

The control of the superficial reflexes varies somewhat from that of the deep reflexes. The disappearance of the superficial reflexes after the cerebral lesion indicates cerebral control; the fact that they do not become exaggerated in disease of the pyramidal tract shows that they have little if any representation in the spinal cord.

The control of the Babinski reflex more nearly approximates that of the deep than that of the superficial reflexes.

The text-book instruction varies regarding the condition of the reflexes in the initial stages of apoplectic hemiplegia from the statement that the deep reflexes are increased, the superficial usually diminished (Osler) to the recognition of lessened reflexes as the usual custom (Oppenheim, Dana). Mills (1897, p. 247) takes, perhaps, the most common view, namely, that while the superficial and deep reflexes may be depressed or absent in the early hours this is not the rule, at least for the latter, and that probably it is most common "that at first the skin reflexes are abolished and the muscle jerks are increased."

As a contribution tending to establish the rule, the following cases coming under our observation during the past year are recorded. It will be seen that out of 13 cases of cerebral apoplexy and cerebral trauma, in 8 there was lessening of reflexes on the paralyzed side; in one additional case the reflex was lessened in the part earliest and most completely paralyzed, and increased in the other paralyzed parts.

In one case, further, of depressed fracture in the lower Rolandic area, there was lessening of the knee-jerk on the paralyzed side and increase of the Achilles reflex with ankle clonus.

In one case, again, the superficial reflexes were absent, the deep exaggerated on the paralyzed side.

In the remaining two cases there was increase of knee-jerk on the paralyzed side. These two cases were first seen 8 and 22 days respectively after the apoplectic attack, so that even here it is by no means certain that the reflex was increased from the first.

The history of the 13 cases is briefly appended.

In the following cases there was *lessening* of reflexes on the paralyzed side.

Case 1. Woman about 65—apoplectic shock some days ago. Right hemiplegia. Knee jerk moderate left, absent right. Achilles active left, moderate right.

Case 2. Man, 63. Apoplectic shock with right hemiplegia. Examined one hour later—unconscious, breathing heavy, flaccid paralysis, arm and leg. Knee jerk average activity left, absent right. Babinski present right. No clonus. Epigastric, abdominal and cremasteric reflexes active left, wanting right. Muscles right or left react perfectly to Faradic current. Twelve hours later, knee jerks present right but very slight. Thirty-six hours after, shock right knee jerk still more active. On the third day about the same. This patient died on the fifth day, the right knee jerk remaining throughout much less active than the left.

Case 3. Child 19 months. Convulsions followed by right hemiplegia. Knee jerk practically wanting on paralyzed side, returned in two weeks.

Case 4. Man, about 20. Embolus, rheumatism, endocarditis and pericarditis. Sudden attack 4 o'clock (embolus) loss of power right arm and leg, flaccidity, at 5:15. Knee jerk absent right, active left. At 7:30 more active right than left. Abdominal and epigastric still absent on right, lively on the left—cremaster and plantar slight on right and active on left. Recovery complete early the next morning, but before 11 he suddenly lost power again in right arm and leg. *The knee jerk was nearly wanting on right and Babinski was present.* Two days later the knee-jerk and cremaster were equal on the two sides, but the epigastric and abdominal had not yet returned on the right.

Case 5. Man, 21, fell 11 feet, striking left parietal region. Right hemiplegia followed. Scalp wound without fracture. Forty-eight hours after injury the knee-jerk was present on both sides but much less active on the right. Plantar reflex of flexor variety alike on both sides. No clonus. Abdominal and epigastric present and alike on both sides. Cremaster present sluggish, both sides alike. Knee-jerk on the right increased daily and on the sixth day after the accident was the same as the left, both being at this time more lively than the left was originally.

Case 6. Man, 35. Struck on head, automobile accident, 2 days ago, unconscious, delirious; left arm and leg paralyzed. Knee-jerk wanting both; Achilles present both but less left; cremasteric lively right, slow left.

In the following case there was bilateral paralysis with absence of reflex on both sides.

Case 7. Woman, 57, fell down stairs, became unconscious with signs of fracture of base (immobile pupils and sunconjunctival ecchymosis). Seen 18 hours after accident: relaxed paralysis complete on left, nearly so on right. Absence of both knee-jerks and of plantar, abdominal and epigastric reflexes. No Babinski.

In the following case there was unilateral paralysis with lessening of superficial reflexes on the paralyzed side and absence of knee-jerk and Achilles on both sides.

Case 8. Man, 33. Rheumatism, heart negative, marked atheroma at wrists and temples. Sudden left hemiplegia two months ago without unconsciousness. Arm, leg and face affected. Complete relaxation of left arm and leg to-day, but all movements possible. Knee-jerk and Achilles absent both; no Babinski; abdominal lively right, very slight left; epigastric lively right, practically wanting left; cremaster slight right, wanting left. The wrist reflexes were lively on both sides especially on the *left*.

In the following case the reflex was *lessened* in the part earliest and most completely paralyzed and *increased* in the other paralyzed parts.

Case 9. Woman, 52, corpulent; probable hemorrhage, left hemiplegia, subacute onset, first arm, then leg; parts said to have been somewhat rigid from first. Seen 21 hours later. Unconscious practically but could be roused enough to answer simple questions; arm relaxed, leg moderately rigid. Wrist reflex present right, faint left; knee-jerk fairly active both sides, more active left (paralyzed side); Achilles slight right, active left; abdominal and epigastric present right, absent left.

In the following case certain reflexes were lost and others exaggerated on the paralyzed side. Those which were lost corresponded to the immediate site of brain injury.

Case 10. Boy, 4 years, hit by automobile at 10 A. M. Depressed fracture over lower Rolandic region; trephined. Unconscious, restless with immobility of right arm, face and leg with flaccidity. Knee-jerk active left, faint right. Achilles normal left, exaggerated right and produces clonus. Babinski right. Patient died that night.

In the following case of incomplete paralysis the superficial reflexes were absent on the paralyzed side, the deep were exaggerated.

Case 11. Woman of 60. Sudden hemiplegic attack 2 days ago, probably embolus; face, arm, and leg affected on left. Today condition spastic, very little voluntary motion in arm or leg. Exaggerated knee-jerk especially left, clonus left, none right;

Babinski left, suggested right. No abdominal or epigastric left, abdominal only on right, slight.

In the following cases the knee-jerk was *exaggerated* on the paralyzed side.

Case 12. Woman, 40 years. Apoplectic shock with left hemiplegia; first examined 22 days later. Knee-jerk present right, fairly active left. No Babinski.

Case 13. Man 40. Hemiplegia left, attack 8 days ago. Knee-jerk active both sides, especially left.

We append the folowing cases of vertebral fracture.

Case 14. A man about 35 years was painting side of vessel when struck on neck by heavy plank; fell 30 feet. Was unconscious for a few minutes. Complete flaccid paralysis of arms and legs including deltoids. Complete anesthesia to second rib, including the arms. Complete absence of knee-jerk, Achilles, also epigastric, abdominal and cremasteric reflexes. No Babinski. All muscles react perfectly to Faradic current. Patient lived two days and a half during which time the condition of the reflexes and electrical reaction remained as at first examination.

Post-mortem examination showed crush of the cord at level of fifth cervical vertebra with rupture and hemorrhage.

Case 15. A man about 40, fell striking his head and bending his back. Complete loss of motion followed in the lower extremities with loss of knee-jerk, Achilles, abdominal, cremaster and plantar excepting that stroking the sole produced extensive and deliberate motion of the great toe downward and inward on each foot (modification of Babinski?) The epigastric reflex was present. There was anesthesia to the umbilicus. Operation (Dr. Mixter) revealed a fracture of the sixth dorsal vertebra.

We append also two cases of uremic coma.

Case 16. Woman, 50. Headache 6 days, restlessness and vomiting at frequent intervals, several days with temporary unconsciousness followed by thickening of speech. Urine high colored, contained albumin and casts; 27 oz. passed in 24 hours. One day complete anuria. Developed Korsakow's condition. On first examination by Dr. Walton there was flaccidity of the limbs with absence of knee-jerks on both sides. No Babinski. The pupils were small. There was cardiac enlargement, atheroma of arteries, and blood pressure of 150, Riva Rocci, 9 cm. cuff. Albuminuric retinitis. Three days later violent headache ensued with two attacks of brief unconsciousness with thickness of speech. Both knee-jerks still absent. Seven days after first examination complete left hemiplegia, rapid onset with relaxed paralysis. Knee-jerks still absent both sides. One month after first examination there was slight motion in fingers and toes. The knee-jerks had returned on both sides, moderate on the right, more active on the left. No Babinski. Improvement continued, greater in the leg. The patient is now up and about.

Case 17. Female, 51. Three days before examination by Dr. Walton developed Korsakow's symptom and before 24 hours became unconscious. Urine contained albumin and casts, and 17 oz. were passed in 24 hours. All extremities flaccid but legs are drawn up on plantar stimulation. Absence of knee-jerk and Achilles both sides. Patient died two days later.

It seems a reasonable conclusion that lessening and loss of reflexes is the rule in apoplexy. It seemes reasonable further to conclude that *complete* paralysis of cerebral origin is accompanied by absence of reflexes both superficial and deep, and that in the exceptional cases of apoplexy in which the reflexes are increased from the outset the withdrawal of cerebral influence has not been at any period complete.

The explanation that coincident disease of the spinal cord has abolished the reflexes is rendered improbable by the number of cases cited and the reappearance of the reflex. Under such a supposition we should have to assume the remarkable coincidence of cord disease limiting itself to the corresponding gray matter, and to assume also a recovery of reflex after such disease contrary to the usual experience.

It is necessary to explain the fact that in the case of cerebral hemiplegia, however complete, the reflexes almost invariably return and become active, while in the complete spinal lesion they do not.

It must be remembered, here, that in the complete spinal lesion both the crossed and uncrossed pyramidal fibers are destroyed, whereas in the case of hemiplegia the uncrossed fibers are still intact, thus furnishing a certain degree of the cerebral element which is requisite to the reflex.

We would also call attention to the not infrequent loss of reflexes in cerebral tumor. Out of 123 cases of tumor analyzed by the writers[3], the knee-jerk was increased in 39, diminished or absent in 32, in 5 of which there was definite statement of hemiplegia with diminution of knee-jerk on the paralyzed side.

The absence of reflex not infrequently observed during an attack of epilepsy, in chorea, in idiocy, and in other forms of cerebral disorder, requires no elaborate explanation when the cerebral element in the production of the reflex is recognized.

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[3] JOURNAL OF NERVOUS AND MENTAL DISEASE, August, 1905.

Such expressions as "irritative inhibition" may surely be dispensed with, as well as the assumption that the brain can exercise increased inhibitory influence at a time when it is deprived of such important functions as consciousness and voluntary motion.

It may well be that the complete scheme of the human reflex mechanism is far more complex than this outline would suggest, and that the reflex arcs at different levels of the brain may possess varying degrees of automatism and activity; this supposition would not be incompatible with the general principle we have formulated.

It is not impossible, for example with regard to the deep reflexes, that the arcs at the level of the red nucleus preside over a reflex less active than that of the lumbar cord, but more active than that of the cortex. In this event destruction of the paths between the cortex and the red nucleus would result in a reflex exaggerated over the normal but not identical with that produced by disease of lateral tracts of the cord.

Conclusions.

The deep reflex is a resultant of the activity of cerebral and spinal arcs, the longer arcs tending to produce a deliberate and moderate reflex, the shorter arcs an active and violent reflex.

The deep reflex varies in healthy individuals and in the neuropsychoses according to the predominant influence of the longer or shorter arcs.

In disease of organic origin the partial withdrawal of the higher influence causes the spinal type of deep reflex, but the complete withdrawal of the higher influence causes abolition of the deep reflexes, since the spinal arc alone is incapable (in man) of sustaining the burden.

Upon re-establishment of the higher influence the reflexes return, the spinal type predominating if the re-establishment is partial, the normal type if it is complete.

Initial lessening or loss of deep reflex in the paralyzed parts is the rule in apoplexy. This condition persists for a period varying from half an hour to a number of days, after which these reflexes become normal or assume the spinal (exaggerated) type according as the return of cerebral influence has been complete or partial.

In the exceptional cases of apoplexy with initial exaggeration of deep reflexes the withdrawal of cerebral influence has been from the first incomplete.

The superficial reflexes, like the deep, have a cerebral control, and disappear on withdrawal of that control. The fact that they do not become exaggerated in disease of the pyramidal tract shows that they have little if any spinal representation.

The control of the Babinski reflex more nearly approximates that of the deep than that of the superficial reflexes.